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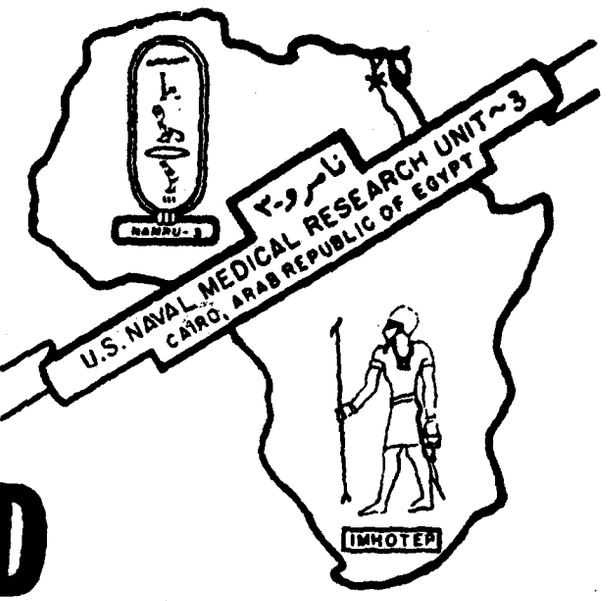

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A REVIEW OF ANAEROBIC INFECTIONS OF THE ORAL CAVITY IN EGYPT

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A Review of Anaerobic Infections of the Oral Cavity in Egypt

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INTRODUCTION

Infectivity associated with anaerobes has been fairly recently detected yet has drawn the attention of many microbiologists. Descriptions of Vincent's gingivitis were reported by the Greek scientist Xenophen in the fourth century B. C. Pasteur discovered obligatory anaerobic bacteria in some diseases that he studied early in his life. Understanding of the disease-causing potential of these fastidious microorganisms did not occur until 1897, when Veillon and Zuber isolated anaerobes from patients with purulent infections [12]. However, difficulties in the cultivation of anaerobes have confined the information of how important these microorganisms are to the field of bacteriologists. Advances in anaerobic culture techniques during the last 20 years showed that these oxygen-sensitive bacteria can cause mixed infections which may involve the central nervous system, head and neck, lungs, abdomen, pelvis, skin, and soft tissues [15].

ANAEROBIC BACTERIA IN THE ORAL CAVITY

Mucous membranes of the mouth and pharynx are often sterile at birth but may be contaminated by passage through the birth canal. Viridans steptococci become established and remain prominent for life. Other microorganisms such as gram-negative diplococci, diphtheroids, and occasionally lactobacilli are added afterwards [10]. When teeth erupt, anaerobic conditions begin to exist in the gingival, crevicular, and interproximal areas. Anaerobic spirochetes, *Bacteroides*, *Fusobacterium*, some anaerobic vibrios, *Actinomyces*, and lactobacilli establish themselves [26]. The oral cavity, as a matter of fact, represents a host environment possessing features that favor the location and growth of a great variety of microorganisms. There are soft and hard structures, and certain areas show differences in oxygen tension and in nutrition. Some surfaces protect the organisms from friction and the flow of oral secretions, whereas other surfaces do not [22].

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Epidemiological studies have demonstrated a strong positive correlation between oral microorganisms and diseases of gums, teeth and oro-facial tissues [1,2]. That dental plaque is the cause of periodontal disease and not a sequelae to the disease was shown by Loe and coworkers [13,10]. Taxonomic and anatomical studies of plaque associated with periodontal health and disease have demonstrated that differences in the microbial populations of plaque may be responsible for the initiation and progression of disease [27]. The consistent isolation of large numbers of anaerobic and capnophilic bacteria from the depths of periodontal lesions has explained the important role played by these organisms in the pathology of the oral cavity [17]. Reports published on the various aspects of these conditions have confirmed the involvement of anaerobes in severe oral infections [4,6].

ANAEROBIC INFECTIONS

Certain characteristics are suggestive of anaerobic infections. Of these, the tendency to have mixtures of organisms and the disposition toward the formation of closed-space infections are very pronounced [10]. Under normal conditions, but when introduced into the surrounding tissues of the host, they become pathogenic based on individual virulence factors which are not shared by all members of the resident microflora [15]. Koch's postulates concerning the monotetiology of diseases are not applicable to anaerobic synergistic infections. In other words, anaerobic infections have generally been considered to be bacteriologically non-specific [14]. They are probably biochemically specific, being dependent on an array of metabolites produced by different members in the infective combination [27].

ANAEROBIC INFECTIONS OF THE ORAL CAVITY

It may be appropriate to discuss these infections according to their origin as odontogenic and non-odontogenic.

Odontogenic infections -

These involve the general inflammatory conditions affecting the periodontium.

1. Gingivitis: Healthy gingival sulci usually harbor some scant microflora that are dominated by gram-positive organisms and include species of *Streptococcus* and facultative *Actinomyces* [26]. Initiation of gingivitis is believed to be a major consequence of the bacteria present in the supragingival plaque. The

later appearance of gram-negative rods and proliferation of anaerobes clearly indicates this sequential process [30,31]. Species of *Bacteroides, Fusobacterium, Hemophilus,* and other gram-negative rods comprised about 45% of the total gingivitis isolates [26]. While microorganisms and their products played a significant role in the inflammatory lesion, a variety of metabolic factors had to determine the host resistance [23,29]. In Egypt, the poor oral hygiene probably explains the widespread gingivitis-associated plaque. In our laboratory, there is evidence to support the view that Egyptians have microflora comparable with old plaque (unpublished data).

2. Periodontitis: Once the disease process involving the gingival tissue extends to include periodontal fiber destruction and loss of alveolar bone concomitant with apical migration of the epithelial attachment, the disease is designated periodontitis [17]. The most common form of periodontitis is chronic in nature and is characterized by the presence of 30-40% gram-positive filamentous organisms, mainly *Actinomyces* [5,28]. Subgingival plaque in this condition harbors many anaerobic gram-negative rods and spirochetes, as documented by electromicroscopy [12]. Predominant microflora inhabiting the base of deep pockets in advanced adult periodontitis constituted an average of 74.3% gram-negative rods that were generally difficult to maintain and identify [25]. Black-pigmented *Bacteroides* (BPB) and *Fusobacterium nucleatum* were the most frequent organisms isolated from this disease.

Rapidly progressing periodontal breakdown in young patients is usually refered to as juvenile periodontitis. Lesions in this case are either confined to permanent first molars and incisors or generalized. According to SLOTS [26], the deep pocket flora in juvenile periodontitis are mainly gram-negative organisms (about 65%) dominated by isolates of *Bacteroides* and other variable oral bacteria that need to be identified. Microorganisms recovered appear to differ from those found in the adult forms of periodontitis [17]. This may suggest a unique microbiology associated with the condition. *Actinobacillus actinomycetem-comitans* has recently been recovered from a large percentage of juvenile periodontitis patients [7,9]. The organism is a micro-aerophilic gram-negative bacillus isolated from patients with actinomycosis [8,20] and may exist as part of the normal oral flora. Further studies are needed to illustrate the exact role that may be played by this microorganism and others in the etiology of juvenile periodontitis.

Non-odontogenic infections

These are initiated by indirect intra- or extra-oral sources.

1. Actinomycosis: Actinomycetes are a heterologous group of filamentous bacteria, the anaerobic speices of which are part of the normal flora of the mouth. Oral actinomycosis is a chronic suppurative disease that spreads by direct extension, forms draining sinuses, and is caused by *Actinomyces israelii* and related anaerobic filamentous bacteria [21]. It is now clear that this organism and others (*Actinomyces viscosus* and *Actinomyces naeslundii*) possess efficient mechanisms to establish themselves at the site of gingival inflammation, and furthermore, they are able to participate in the activation of host reactions assumed to be involved in tissue destruction [11].

2. Abscesses: Many cases of dental abscesses present annually worldwide. Such abscesses are either associated with frank dental carious lesions and pulp tissue exposure or with advanced periodontal disease [18]. However, some are without obvious antecedent pathology [24]. Historically, bacteria of the viridans *Streptococcus* group have been thought to be the major organisms in periapical abscesses. Recent studies incorporating anaerobic sampling and culture techniques have reported a greater number of mixed infections and larger populations of bacteria [34]. In Egypt, it appears that combinations of BPB, *Veillonella*, and *F. nucleatum* are rather frequent in the samples collected from dental abscesses. Incidences of completely anaerobic abscesses were comparatively low in this region [33]. Evidence from our laboratory and findings of others [18,34] suggest an efficient synergistic activity between most microbial combinations identified in oral abscesses.

USE OF ANTIBIOTICS

Once anaerobic infections have been established, the lesions become favored by reduced blood supply, formation of necrotic tissues, and low oxidation-reduction potential, all of which can interfere with the administration of antibiotics [17]. Also, anaerobiosis in mixed infections has a deliterious effect on the function of aminoglycoside antibiotics (streptomycin, neomycin, kanamycin, gentamicin, and others) which require oxygen-dependent transport across the bacterial cell wall [3]. The high number of bacteria, low pH, organic acids, and divalent cations in abscess cavities act to reduce the activity of antibiotics [15]. Many organisms have developed resistance to the antibiotics in common clinical use. Those which produce β-lactamase, for instance, are able to destroy the effictiveness of β-lactam antibiotics [19]. In Egypt, it is assumed that the widespread use of non-prescription antimicrobial agents has affected the susceptibility of the oral organisms involved in any disease process.

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